83 Indigestion and Heartburn

HOWARD SCHACHTER

Definition

Indigestion is a term commonly used by patients and physicians to indicate some form of gastrointestinal tract upset. It includes a wide variety of symptoms, which in turn may be manifestations of a wide variety of gastrointestinal and nongastrointestinal diseases. Often these symptoms prove to be a reflection of gastroduodenal dysfunction secondary to stress. The words *indigestion* and *dyspepsia* may be used interchangeably, usually to describe one or more symptoms experienced shortly after eating, simply implying a disorder of the digestive processes. Among such symptoms are postprandial nausea and occasionally emesis; upper abdominal bloating, fullness, or discomfort; belching and flatulence; and, less commonly, a bad taste in the mouth, a coated tongue, fatigue, somnolence, or headache.

Occasionally patients use the term *indigestion* to describe the symptoms of *heartburn;* heartburn, however, is defined more specifically as a substernal burning sensation of variable intensity that may extend toward the neck or base of the throat.

Technique

Understand the patient's symptoms. What does the patient mean by the terms indigestion and heartburn? If the patient complains of indigestion, specifically which symptoms are present? Is there postprandial nausea? If vomiting has occurred, what is the nature of the vomitus? What is its volume? Is it acidic? Is it bilious? Does it contain undigested food? Does it contain fresh or altered blood? Does it have other unusual characteristics? Does the patient have upper abdominal bloating, fullness, or discomfort? Are belching and flatulence prominent? Does the patient complain of a bad taste in the mouth or coated tongue? Has the patient experienced fatigue, somnolence, or headache? Does the patient have heartburn but call it indigestion?

After the symptoms have been clarified, inquiry should be directed toward an understanding of their genesis. When did these symptoms begin? Are they constant or intermittent? What precipitates them? What affords relief? If intermittent, how long do they last? What pattern, if any, do they show? Do they follow meals? Do they occur after specific food or drink? Do they relate to taking some new medication? Do they occur with activity? Do they change with position? Do they relate to bowel activity? If pain is present, what is its location and intensity?

What are the patient's dietary habits? Are meals skipped? Are suppers unusually large? Does the patient drink excessive alcohol, coffee, or cola drinks, or smoke cigarettes? Does the patient chew his food well, or is he or she edentulous? Does the patient lie down soon after eating? Are the meals eaten "on the run" or in a relaxed manner? Are there ongoing unusual, stressful life situations or emotional conflicts? Are symptoms associated with identifiable stressful situations?

With respect to indigestion, inquire about symptoms of, and search for signs of, a variety of gastrointestinal and extragastrointestinal disorders because the differential diagnostic considerations are extensive. With heartburn, inquire about occasionally associated acidic or bilious regurgitation, dysphagia, odynophagia, and chest pain, the last being a symptom of diffuse esophageal spasm. In patients having complaints of indigestion and/or heartburn, thorough inquiry into underlying psychosocial factors is of major importance because emotional stress and conflict frequently prove causative. Symptoms may be augmented further by the patient's anxiety about his or her physical well-being. Only after appropriate diagnostic studies have reassured the patient (and the physician) can both parties begin to deal effectively with an underlying emotional basis for the patient's symptoms.

Basic Science

Postprandial upper abdominal fullness may result from excessive intake of food or drink, especially of carbonated beverages, but the complaint of indigestion more likely is the consequence of *aerophagia* with gastric distention or of *delayed gastric emptying,* the latter secondary to mechanical or motor disturbance. Occasionally, large esophageal diverticula, esophageal strictures (malignant or benign), or esophageal motor disturbances as seen in achalasia or scleroderma may result in similar postprandial fullness, which may be described as indigestion. Preceding gastric resectional surgery for the treatment of gastric malignancy or peptic ulcer may significantly reduce gastric capacity, again resulting in postprandial fullness or indigestion. Associated bilious reflux also may be symptomatic.

The stomach can be thought of as two functional units: the proximal reservoir (fundus and body) and the distal pumping area of mixing and emptying (antrum). The pylorus is a low-pressure sphincter that prevents large particles of food from exiting; it also prevents reflux of duodenal content into the stomach. Liquids exit the stomach faster than solids. Delivery of gastric content into the duodenum is dependent on the presence of a 3 cycle per minute electrical control activity, with periodic antral contractions occurring in response to a second electrical event known as the electrical response activity. The entire process is governed by complex neural and humoral factors.

The rate of gastric emptying may be inhibited by meals of high osmotic pressure or high fat content, with inhibitory receptors probably located in the duodenum and proximal jejunum. Studies of Hunt and Stubbs (1975) have yielded data indicating that nutritive density (kilocalories per milliliter) rather than initial volume determines the rate of gastric emptying. Isocaloric amounts of fat, protein, and

carbohydrate apparently cause equal slowing of gastric emptying. Inhibitory activity is mediated by neural and hormonal mechanisms, and, presumably, by their interactions. Metabolic products of ingested food also may play a role. The relative importance of neural and hormonal factors in the regulation of gastric emptying remains uncertain. The gastrointestinal hormones gastrin, secretin, and CCK-PZ all inhibit gastric emptying. GIP (gastric inhibitory peptide) and VIP (vasoactive intestinal peptide) also retard gastric emptying, whereas motilin increases gastric motility. Enterogastrone, a crude intestinal extract liberated from the duodenum by micellar fat exposure, also inhibits gastric emptying. Sympathetic, vagal, and other inhibitory gastric innervation are involved in retarding gastric emptying. Examples of diseases in which mechanical pyloric obstruction accounts for delays in gastric emptying include tumors, peptic ulcer disease, and idiopathic hypertrophic pyloric stenosis.

Examples of conditions in which acute or chronic delays in gastric emptying are observed in the absence of mechanical obstruction are given in Table 83.1. The causes of the impaired gastric emptying in many of these conditions remain incompletely understood. Gastric emptying may be altered variably by vagotomy and a variety of surgical procedures, which sometimes lead to dumping, but occasionally lead to chronic gastric retention with nutritional compromise and occasional formation of bezoars. Atrophic gastritis, gastric ulcer, and gastric cancer sometimes are associated with delayed gastric emptying by mechanisms that are obscure. Medications of a wide variety cause symptoms of indigestion by presumed direct gastric irritation (e.g., acetylsalicylic acid and other nonsteroidal anti-inflammatory drugs), by central effects (e.g., morphine), and by anticholinergic effects or side effects (e.g., atropine, various antispasmodics, and phenothiazines).

Bloating, one of the common complaints of the patient with indigestion, may be attributable to gastrointestinal gas. The latter is derived from swallowed air and from gut bacterial metabolism. Postprandial bloating and belching often are related to aerophagia, sometimes a habit of anxious individuals, occasionally aggravated by the ingestion of carbonated beverages or by gum chewing. Complaints of bloating and flatulence are common in patients with the irritable bowel syndrome, apparently the result of an underlying motility disturbance and decreased pain threshold and not the result of qualitative or quantative differences in intestinal gas formation in comparison to normal controls, as clarified by Lasser, Bond, and Levitt (1975).

Table 83.1
Nonmechanical Causes of Gastric Retention

Causes	Examples
Pain or trauma	Biliary or renal colic, migraines, postsurgical state, following traumatic injury to urinary tract, following fractures, following splenic rupture
Infection or inflammation	Gastric ulcer (nonobstructive), acute pancreatitis, appendicitis, peritonitis, sepsis
Immobilization	Paraplegia, postsurgical state
Metabolic	Diabetic ketoacidosis, hypercalcemia, hypocalcemia, hypokalemia, uremia
Neural	Diabetic autonomic neuropathy, previous vagotomy, brain tumor
Drugs	Morphine, anticholinergic drugs, drugs with anticholinergic side effects (e.g., phenothiazines)

Heartburn, the most frequent symptom of gastroesophageal reflux, is experienced variably in response to esophagitis caused by the reflux of irritant acid peptic or, occasionally, alkaline gastric or gastroduodenal content. Presumably such reflux is most often the consequence of an incompetent lower esophageal sphincter (LES), a manometrically demonstrable segment of high pressure normally maintained at the junction of the esophagus with the stomach. Factors influencing gastroesophageal reflux and its effects include the competency of the LES, the volume of gastric content, the quantity and corrosiveness of the refluxed material, the clearance of this material by the esophagus, and local esophageal tissue resistance. Delayed gastric emptying also may contribute to symptoms of gastroesophageal reflux in some patients. Patients may experience increased heartburn after overeating or after recumbency following meals, after eating spicy foods, fatty foods, or chocolate, after drinking citrus juices, cola drinks, coffee, or alcohol, or after smoking. Some of the preceding cause a reduction in lower esophageal sphincter pressure, with presumed consequent increased gastroesophageal reflux. Anticholinergic medications may have similar effects. While alkaline reflux esophagitis may be observed in some patients without preceding gastric resectional surgery, such operations as well as gastroenterostomy particularly predispose to this problem.

Clinical Significance

A sense of fullness, bloating, or upper abdominal discomfort and distention after meals, or, in common parlance, indigestion, suggests aerophagia and/or impaired or delayed gastric emptying. The former usually is a nervous habit; the latter usually is the result of mechanical pyloric or gastric outlet obstruction or is secondary to a multitude of nonmechanical causes, including emotional factors.

Delay in gastric emptying may be evidenced by obvious gastric distention on physical examination or plain abdominal x-rays, by emesis of undigested food many hours after its consumption, or by apparent retained food in the stomach as noted at the time of upper gastrointestinal barium radiographic studies or upper endoscopy. It may be assessed by means of radiopaque meals or by intubation sampling or isotope studies.

The most common causes of mechanical *pyloric or gastric outlet obstruction* are tumors (polyps, carcinoma), narrowing secondary to acute inflammation and edema or chronic scarring from peptic ulcer disease, and idiopathic pyloric hypertrophy. Other neoplastic or infiltrative processes involving the stomach primarily (e.g., carcinoma, gastroduodenal Crohn's disease) or secondarily (e.g., pancreatic carcinoma) also may cause gastric outlet obstruction. The most common causes of nonmechanical gastric retention include both gastrointestinal and extragastrointestinal conditions such as those listed in Table 83.1. Diseases as diverse as scleroderma and acute gastroenteritis also may result in delayed gastric emptying. The other and perhaps largest group of patients with indigestion has anxiety and stress as the chief determinants of this so-called psychophysiologic gastrointestinal reaction. In this group, extensive clinical evaluation yields no apparent organic explanation for symptoms.

Heartburn usually is the result of acid or alkaline gas-

troesophageal reflux esophagitis. Gastroesophageal reflux can be demonstrated by means of barium studies, by manometric placement of a pH probe, or by radioisotope study. When the patient gives a classic description of heartburn, further study rarely is needed. When one is uncertain of the source of the patient's symptoms, the Bernstein test can be done. Briefly stated, this test monitors the patient's response to acid perfusion of the esophagus. This test often is helpful, but by no means is it always conclusive.

Endoscopic findings of esophagitis run the gamut from imperceptible grossly, to erythema, to frank erosions and exudates, and even to ulceration and stricture formation. Suction biopsies of esophageal mucosa may demonstrate thickening of the basal layer and elongation of the dermal pegs which may extend nearly to the mucosal surface. Manometric measurements of the lower esophageal sphincter tend to be low in most patients with gastroesophageal reflux; however, some patients with normal pressures may have symptoms of reflux, and some patients with low pressures may not. As in the case of many patients with indigestion, extensive studies in patients complaining of heartburn also may prove unrevealing. Nevertheless, such comprehensive evaluation may serve as the basis of an effective therapeutic approach to the large group of patients whose symptoms are emotionally based.

Coronary artery disease producing angina is often described by patients as "indigestion," "heartburn," or a sense of "fullness." A valuable clue is the association of the duration with exertion and its relief with rest. See Chapter 9, Chest Pain or Discomfort.

References

Alvarez WC. Nervousness, indigestion and pain. New York: Paul B. Hoeber, 1943.

Dodds WJ, Hogan WJ, Helm JF, et al. Pathogenesis of reflux esophagitis. Gastroenterology 1981;81:376–94.

*Hogan WJ, Dodds WJ. Gastroesophageal reflux disease (reflux esophagitis). In: Sleisenger MH, Fordtran JS, eds. Gastrointestinal disease. Philadelphia: W.B. Saunders, 1989; 594–619.

Hunt JN, Stubbs DF. The volume and energy content of meals as determinants of gastric emptying. J Physiol (London) 1975;245:209–25.

Lasser RB, Bond JH, Levitt MD. The role of intestinal gas in functional abdominal pain. N Engl J Med 1975;293:524–26.

McCallum RW. Motor function of the stomach in health and disease. In: Sleisenger MH, Fordtran JS, eds. Gastrointestinal disease. Philadelphia: W.B. Saunders, 1989;675–713.

McCallum RW, Berkowitz DM, Lerner E. Gastric emptying in patients with gastroesophageal reflux. Gastroenterology 1981;80:285–91.

Richter JE, Castell DO. Gastroesophageal reflux. Ann Intern Med 1982;97:93–103.

Thomas JE, Baldwin MV. Pathways and mechanisms of regulation of gastric motility. In: Code CF, ed. Handbook of physiology. Washington, D.C.: American Physiological Society, 1968;4:1937–68.

Wolf S, Wolff HG. Human gastric function. New York: Oxford University Press, 1943.